Current Status of Immunoglobulin E (IgE)

WHILE QUANTITATIVE measurements of serum IgE levels are not diagnostic of the allergic state, the great majority of persons with elevated IgE levels manifest allergic symptoms. A rare individual with low serum ige may have proven allergy. In addition, serum ige levels have been shown to be greatly elevated (thousands of ng* per ml) in parasitic infestations. The mean serum ige level in non-atopic persons as determined in several laboratories varies from 143 ng per ml to 248 ng per ml with ranges (in non-atopic adults) from 19 ng per ml to 950 ng per ml (mean ± 2 standard deviation).

Serum IgE is unique in having the widest range of all the immunoglobulins. Levels between 2 and 36,000 ng per ml have been reported. The half-life of serum ige is about two and a half days in contrast to cell-bound ige which persists for several weeks.

ige does not normally cross the placenta and most newborns do not produce significant quantities of Ige. The lymph nodes of the gastrointestinal and respiratory tracts appear to be the principal areas of IgE production. Maturation results in increasing IgE levels throughout childhood paralleling IgA development. At all ages there is wide variation in IgE levels. In allergic children (with ragweed hay fever) the serum ige level was found to be inversely related to age of onset.

Although the serum IgE level did not correlate with the symptom index in ragweed hay fever patients, a small rise in serum ige was noted during the ragweed season in untreated persons. After hyposensitization therapy this rise did not occur.

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Alpha-1 Antitrypsin Deficiency

NORMAL HUMAN SERUM inhibits the activity of proteolytic enzymes. Approximately 90 percent of this activity as measured by trypsin-inhibiting capacity is attributed to the alpha-1 globulin fraction of the serum and is termed alpha-1 antitrypsin,

Inherited deficiency of alpha-1 antitrypsin is associated with a high frequency of early onset, lower lobe emphysema which might be the result of the autodigestion of pulmonary tissue by the breakdown products of white blood cells and alveolar macrophages. Alpha-1 antitrypsin deficiency should be considered in the differential diagnosis of bronchial asthma and other chest diseases of young adults.

Whether slight to moderate deficiency of alpha-1 antitrypsin predisposes to chronic obstructive lung disease is still unresolved.

The diagnosis is made by finding a low level or absence of alpha-1 globulin on paper electrophoresis and confirming this by measurement of the serum trypsin-inhibiting capacity by chemical or immunological means.

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Disodium Cromoglycate in the Treatment of Bronchial Asthma— **Further Clinical Trials**

Further investigation of disodium cromoglycate (cromolyn sodium) has confirmed previous reports of its efficacy in the treatment of bronchial asthma. (See Epitomes of Progress, Calif Med 112:40, June 1970.) The drug is administered as

^{*}ng: nanogram or 10-9 gram